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"AN EXPERIMENTAL MODEL FOR CLOSED HEAD IMPACT INJURY"

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The use of cadavers in head injury research can be advantageous because of the normal skull anatomy present and the normal size and shape of the intracranial contents. Certainly the postmortem brain undergoes autolysis, CSF dynamics are absent and vessels are lax. The latter, however, still have enough integrity within a couple of days of death to not allow red blood cells to escape from the vascular lumen into the brain parenchyma. We hoped to utilize this observation to serve as a basis for repressurization of the vascular system (as well as the CSF space) to more closely approximate the living condition. By this method, the results of experimentally induced injury using the cadaver might more closely approximate those anticipated or seen in the living traumatized patient.

As a baseline (four specimens), the carotid and vertebral arteries and jugular veins were cannulated in the neck of refrigerated cadavers less than 48 hours from death. Following the wash-out of the intracranial circulation using normal saline delivered to the carotids and vertebrals at 150 mm/Hg pressure, a mixture of India ink in normal saline was perfused into the same vessels under the same pressure over a period of 30 minutes. Catheters were also palced in the cerebral ventricles via twisted drill craniotomies and the ventricular pressure maintained at 70 mm of saline above the forehead in the supine patient over the same

time period as the vascular perfusion. At the end of the perfusion time, the vascular perfusate is changed to an India ink/formalin solution for five minutes.

Following this, a forensic autopsy is performed on the intracranial contents. We noted variable perfusion of the brain which is assayed by the blue-black hue that a well perfused brain demonstrates. This variable perfusion is probably related to anatomical variations in the circle of Willis. In well perfused regions, 3 mm sections were taken, further fixed in 10 percent formalin, then passed through absolute alcohol and cleared with methyl salicylate. This treatment renders the brain substance translucent and allows visualization under the light microscope of the gross vascular anatomy. We can clearly visualize arterioles and capillaries in these preparations and it is apparent that the India ink in well perfused areas penetrates even the small vessels and does not extravasate into the brain parenchyma. The only exception to this finding in the control studies is noted where the ventricular catheters penetrated the cerebral substance. In these regions, extravasation of India ink grossly into the subarachnoid space (presumably from vessels ruptured when the tube is placed), and on salicylate cleared sections under the light microscope, into the cerebral parenchyma, documents the potential sensitivity of this method in detecting vascular trauma. Such vascular trauma plays an extremely important role in brain damage following human head injury.

In the more experimental situation, the same vascular and CSF pressurization is accomplished plus the placement of intracranial pressure

transducers and the cadaver heads are then injured using a dropped weight or sled technique. Once again, forensic autopsies were conducted on the head. In well perfused specimens, we have noted extravasation of India ink in many cerebral areas including the subarachnoid space, the vertebral cortex and white matter and occasionally the midbrain. Most of these areas of extravasation are visible to the naked eye (all are corroborated microscopically) and bear a reasonable resemblence to the cerebral contusions and subarachnoid hemorrhage seen in human clinical material, either at craniotomy or autopsy.

Although the numbers of experimentally induced injured cadavers is small (10) there is a suggestion of increasing amounts of vascular damage with increasing amounts of force delivered to the head.

With the advent of computerized cranial tomography for use in the living head injured patient, many cerebral contusions and hematomas are visualized that before could only be inferred. There is certainly some similarity between these clinical contusions and the "lesions" we cause experimentally, suggesting the potential relevant information that may be gained regarding threshold forces to produce human intracranial vascular injury and the interfaces that may be necessary to prevent it.

Certainly a number of problems exist within this technique. The variability of the perfusion needs further work and we are developing a perfusion system using a vibratory input that may result in more complete perfusion irrespective of vascular anatomical variations. More thorough perfusion is required before we can get any kind of good assay of total cerebrovascular injury in these cadavers.

Doctor A. K. Ommaya has made the pertinent comment that we may also be creating an edematous brain by this perfusion technique as saline leaks from the vessels into the brain parenchyma. Certainly trauma to an already edematous brain may be confusing in the ultimate injuries produced. I have not been impressed with the gross edematous nature of the brains, but light microscopic and possibly even electron microscopic sections should answer this question.

We look forward to reporting in the future on our further uses of this technique.